IN BRIEF

NEUROLOGICAL DISORDERS

Large recurrent microdeletions associated with schizophrenia

Stefansson, H. *et al. Nature* 30 Jul 2008 (doi:10.1038/nature07229)

Rare chromosomal deletions and duplications increase risk of schizophrenia

The International Schizophrenia Consortium *Nature* 30 Jul 2008 (doi:10.1038/nature07239)

The genetics of complex mental diseases are poorly understood, partly because of negative selection pressure on risk alleles. Now, two independent large-scale genome-wide studies confirm previously identified genetic associations with schizophrenia and discover some new ones. Both studies identified spontaneous copy-number variants and deletions on chromosomes 1 and 15 that are linked to increased risk. Furthermore, one study found a greater overall frequency of copy-number variation in the genome of patients with schizophrenia. These approaches might lead to the identification of additional and more-prevalent risk variants for the disease.

NEURONAL CIRCUITS

Synaptic release of GABA by AgRP neurons is required for normal regulation of energy balance

Tong, Q., Ye, C.-P., Jones, J. E., Elmquist, J. K. & Lowell, B. B. *Nature Neurosci.* 10 Aug 2008 (doi:10.1038/nn.2167)

Hypothalamic Agouti-related protein (AgRP)-expressing neurons, which also release neuropeptide Y and the inhibitory neurotransmitter GABA (γ-aminobutyric acid), are critical regulators of feeding behaviour and body weight. A new study highlights the importance of GABA release from these neurons in regulating energy balance: mice lacking the vesicular GABA transporter specifically in AgRP neurons were lean and resistant to diet-induced obesity owing to an increase in energy expenditure. Furthermore, they were resistant to the effects of the appetite-stimulating hormone ghrelin, suggesting that GABA neurotransmission in the hypothalamus also mediates the orexigenic effect of this hormone and could represent a novel target for treating obesity.

DEVELOPMENT

A nucleostemin family GTPase, NS3, acts in serotonergic neurons to regulate insulin signaling and control body size

Kaplan, D. D., Zimmermann, G., Suyama, K., Meyer, T. & Scott, M. P. *Genes Dev.* **22**, 1877–1893 (2008)

The mechanisms through which the CNS regulates body size, by integrating information about the energy status of the organism and environmental conditions with developmental growth programmes, are largely unknown. Here, the authors showed that the evolutionarily conserved GTPase NS3 acts in serotonergic neurons to regulate body size in *Drosophila melanogaster*. NS3-mutant flies are smaller than wild-type flies, and restoring NS3 expression in serotonergic neurons alone rescued the global growth defect. The authors also found that NS3 acts as an upstream regulator of insulin signalling, which is known to regulate body size, suggesting a potential mechanism through which the nervous system can influence cell size and number.